POSSIBLE CARDIAC BEHAVIOR IN THE ABSENCE OF GRAVITY

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POSSIBLE CARDIAC BEHAVIOR IN THE ABSENCE OF GRAVITY

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ABSTRACT

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The authors study the oxygen cardiac-energy

equilibrium under various conditions. Methods for

preventing and correcting cardiac deterioration in

the absence of gravity are discussed. *author*

Chapter I

Open Oxygen-Energy Equilibrium in Cardiac Fibers

Open equilibrium represents a particular case of equilibrium.   /1*

Equilibrium is produced in this case due to the equality between two

flows - one of which enters the system, while the other leaves the system.

Therefore, open equilibrium will always exist when:

Input flow = K x output flow and K = 1.

This equality makes it possible to study the behavior of a given pro-

cess in a general form. When K = 1 in this study, the process will be in

open equilibrium.

It is assumed today that the energy required for cardiac contraction

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* Note: Numbers in the margin indicate pagination in the original
         foreign text.

is supplied by the ATP which the cardiac cells contain. The utilization of ATP for muscular contraction has been investigated experimentally (Ref. 2), which reinforces this report to a considerable extent. The ATP which the heart contains is primarily synthesized by utilizing the oxygen transported to the heart by blood circulating in the coronary artery.

The ATP employed in cardiac contractions can also be synthesized by means of anaerobic reactions. But these anaerobic reactions produce substances whose exact chemical composition is not known, and we shall assume that they are toxic for inhibiting muscular contractions. It is only by means of oxygen intervention that these toxic substances are burned up or are resynthesized into glucose. Due to the fact that the energy balance of a closed chemical cycle is zero, the only anaerobic energy available is that resulting from the degeneration of the glucose portion which is not resynthesized. Assuming that it is necessary to avoid the accumulation of the toxic substances mentioned above, the heart can only employ anaerobic energy to the extent that it can utilize oxygen for burning up the given toxic substances (in terms of energy, the oxygen consumed to resynthesize the glucose is approximately balanced by the anaerobic degeneration of the same amount of glucose).

On the basis of the statements given above, the ATP which is synthesized by means of anaerobic reactions only constitutes a limited portion of the total amount of ATP which the heart can supply. When the anaerobic production of ATP exceeds this limit due to circulatory conditions, toxic substances accumulate owing to which this type of cardiac operation is only possible during limited intervals of time. A relatively old investigation presented the hypothesis that when the heart expels large amounts of blood

and energy, it contracts a certain type of "debt". O. Lowsley (Ref. 3) found that after consistent exercising on a road six miles long, both the pressure and the pulse remained 20% below the normal values for between 20 to 90 minutes after the exercise.

More recent investigations of circulatory shock have shown that the /2 oxygen debt which the tissues and the heart can contract is limited (Ref. 4, 5). The limit of the oxygen debt which an animal can incur per Kg of tissue without dying increases when digital is administered, as was demonstrated experimentally by J. W. Crowell (Ref. 5). Since digital influences the heart in a specific manner, this fact shows that one portion of the total oxygen debt is incurred by cardiac muscles. When this oxygen debt of the myocardium reaches a certain value - which is independent of the rate at which this oxygen debt originates (Ref. 4) - the heart begins to deteriorate, rapidly leading to death. In the case of recuperation, the oxygen debt is paid (Ref. 4).

Let us now consider the following equality, in which both elements pertain to the same interval of time.

$\underline{a}.\underline{b}$. grams of O_2 consumed by the heart = K. cardiac work (I), where $\underline{a}$ is the mechanical equivalent of energy which can be supplied by the chemical combination of oxygen. The value of $\underline{a}$ can vary somewhat depending on the substrata which are being oxidized (glucose, lactate, fat, some acetonic bodies). With respect to $\underline{b}$, it represents the output of energy supplied by oxygen consumption. This "energy which is supplied" represents not only energy supplied by the chemical combination of oxygen, but also includes energy which is obtained anaerobically due to this oxygen consumption.

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A value of K<1 indicates that the heart is operating at the expense of an excess of anaerobic reactions. In accordance with the experimental data which we obtained, this is only possible in a limited interval of time, which is followed by another interval in which K>1 (this represents the interval in which the oxygen debt is paid). The heart can pay this debt by means of a high oxygen consumption with normal operation, or by means of a normal oxygen consumption with less work. The observations given in (Ref. 3) coincide with the last possibility. In both cases K>1 holds, but for different values of cardiac work. Therefore, the heart can pay an oxygen debt incurred during an exertion, although the values of pressure, pulse, and volume per minute have normalized after this exertion.

A value of K>1 which is maintained for a long interval of time would indicate that the heart can accumulate a very large reserve of free energy, in the form of ATP or substances whose separation produces ATP. However, as is evidenced by cardiac behavior when intoxicated with monoidoacetic acid (Ref. 6, 7), if the anaerobic source of energy which the heart supplies is suppressed - the anaerobic separation of glucogen into lactic acid, which is not possible in the presence of monoidoacetic acid - the heart grows weak after a few beats in the absence of oxygen. As a consequence, a /3 value of K>1 is only possible during a limited interval of time, when the oxygen debt is paid or when a small reserve accumulates which will be used at the first opportunity. Therefore, if the oxygen consumption increases without an increase in the cardiac work, it will indicate that b has decreased.

As a consequence, when larger time intervals are considered, the average value of K will approximate 1. Therefore, (I) represents open

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equilibrium.

Due to the possibility that the heart may incur or pay an oxygen debt slowly (Ref. 4), the interval under consideration should be sufficiently long (several hours). An interval of several minutes is short in relation to this equilibrium.

It is apparent that open equilibrium (I) can be established for a wide range of values for cardiac work, due to the possibility that the organism may vary b and the oxygen contribution. The latter may increase due to coronary vasodilation, an increase in blood pressure (especially diastolic), and an increase in the artery-vein difference in the coronary blood. Although the possibilites of conserving the oxygen-energy equilibrium for different values of cardiac work are extensive, they are not unlimited. As a consequence, there is a maximum limit for the work which the heart may realize per unit of time, in O_2-energy equilibrium. If cardiac work is continuously increased beyond these limits, due to general circulation requirements, the heart will incur an oxygen debt which should be paid quickly to avoid destroying its contractile capability.

Therefore, two limits must be distinguished with respect to the work (always expressed per unit of time) which the heart can perform. One of these limits is the maximum work which the heart can perform but which, as is known, cannot be maintained for a very long period of time (Ref. 7, 8). The other limit is the maximum work which the heart can perform in oxygen-energy equilibrium - i.e., in permanent form. Both limits will naturally depend on the possibilities for adaptation by the organism.

It is not possible to determine the maximum work in energy equilibrium

from the curves showing the cardiac work as a function of oxygen consumption, because it is not possible to know the extent to which these curves depend on an excess of anaerobic reactions. It should be pointed out that all of the preceeding considerations do not depend on a knowledge of the particular metabolic procedures which exist in each one of the processes described for $K<1$, $K = 1$ or $K>1$. These considerations depend only upon the fact that at least a certain proportion of the work performed by the heart must come in the last analysis from the oxygen consumption, in order to maintain the functional integrity of the heart.

Chapter II

Influence of the Decumbent Position on the Cardiac Oxygen-Energy

Equilibrium in a State of Wakefulness

In order to analyze these changes which take place in cardiac oxygen- /4 energy equilibrium when the conditions surrounding the body are changed, it is necessary to study the occurrences in both elements of the equality (I). With respect to the oxygen supply, this depends on the ruling conditions in the coronary circulation.

In the decumbent position, coronary circulation is changed by the following factors. The initial pressure increase in the presso-receptors of the carotid sinus (Ref. 9), which occurs in the decumbent position, causes sympathetic inhibition and parasympathetic excitation. The combined action of these effects causes the heart to slow down, accompanied by coronary vasoconstriction which impedes the coronary flow. Due to this inhibition of the heart, the pressure decreases - which increased initially - in the carotid artery. It is due to this fact that the final pressure change

which occurs in the carotid - when an erect position is changed for a decumbent position - is at a minimum in normal subjects (Ref. 10).

The same reflex reaction starting from the carotid sinus causes a decrease in the peripheral resistance. In a similar manner, in the re-clining position (Ref. 11) the volemia*increases about 15%. In order that a volemia increase of 15% only causes small changes in the cardiac output, a considerable degree of stress-relaxation should occur. If there were no stress-relaxation, the cardiac output would increase by about 100%, if we extrapolate the data obtained for dogs (Ref. 12) to the case of man.

Stress-relaxation does not only occur in the veins (Ref. 13), but very probably also occurs within the tissues (Ref. 14), including the shunts and capillary circulation. The peripheral resistance decreases due to the stress-relaxation. As a consequence of the peripheral resistance decrease - caused by stress-relaxation and by the carotid reflex - the diastolic arterial pressure decreases (Ref. 9), which also results in im-peding the coronary flow.

An increase of 15% in the volemia causes a 13% decrease in the concen-tration of sanguine hemoglobin, the compensation for which would require a relatively long period of time. Therefore, the maximum amount of oxygen which can be transported to the heart will increase in conformity with the coronary flow.

All of these changes pertain to the first element of the equality (I). With respect to the second element, the volume per minute increases by about 23% (Ref. 15), with which the cardiac action is increased in spite

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*Translator's note: This term designates volume of blood.

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of the decrease in the diastolic pressure. Due to the fact that this
increase in the volume per minute involves an increase in the venous re- <u>/5</u>
turn, the coronary flow tends to be considerably increased due to vagal
action (Ref. 10).

If we balance the modifications which both elements of (I) undergo,
we can see that there are both factors of decompensation and of compensa-
tion for the given equilibrium. The decompensation factors reside in the
obstruction of coronary circulation, the obstruction of the oxygen which
it transports, and in the increase of cardiac action. On the other hand,
one compensation factor is the vagal reflex due to an increase in the venous
return.

As a consequence, the decumbent position can initially produce an
effective unevenness between both elements of (I), which would result from
the balance between the mentioned factors of compensation and decompensa-
tion. We shall call the algebraic difference between both elements of (I)
the "unevenness".

Since the cardiac action must be absolutely in equilibrium with the
oxygen contribution, this initial unevenness will be rapidly compensated for
by the regulations governing the body.

It could be argued that this line of reasoning is teleological. But
if, as cybernetics states (Ref. 1), complex systems behave in a teleological
form, we must choose between adapting our form of thinking to this behavior,
or vice-versa. A more extensive knowledge of the structure of the system
could very likely make this teleological manner of thinking superfluous.

Chapter III

General Possibilities for Controlling the Cardiac Oxygen-Energy

Equilibrium

If we analyze the case which was studied in the preceding chapter in

a general form, we shall see that a given disturbance - the decumbent posi-

tion - causes factors of decompensation and of compensation for a given

open equilibrium. The decompensation factors produce an unevenness between

both elements of the open equilibrium, while the compensation factors tend

to reduce said unevenness. If a total resultant unevenness is produced,

this will be compensated by the body controls. Therefore, the total re-

sultant unevenness cannot be measured, because - when it begins or tends

to be produced - it is hidden and compensated for by the body controls.

We shall use the term virtual unevenness to designate the unevenness

between both elements of (I) which will be produced if the body controls

did not come into operation when faced with a given disturbance of the

equilibrium (I).

In accordance with the value of the virtual unevenness, there are

three possibilities in general in relation to the cardiac oxygen-energy

equilibrium (Figure 1). In the three cases, an initial oxygen debt will

be produced, due to the time required for the body controls to be put

fully into operation. If the value of the virtual unevenness is lower than

a certain critical threshold value - once this initial oxygen debt, which

can be very small, is acquired - a stationary state will be established,

which will continue as long as the disturbance endures. The effects which $\underline{/6}$

can be observed as a result of a prolonged decumbent position, with most

of the time spent in a state of wakefulness, behave as if they were

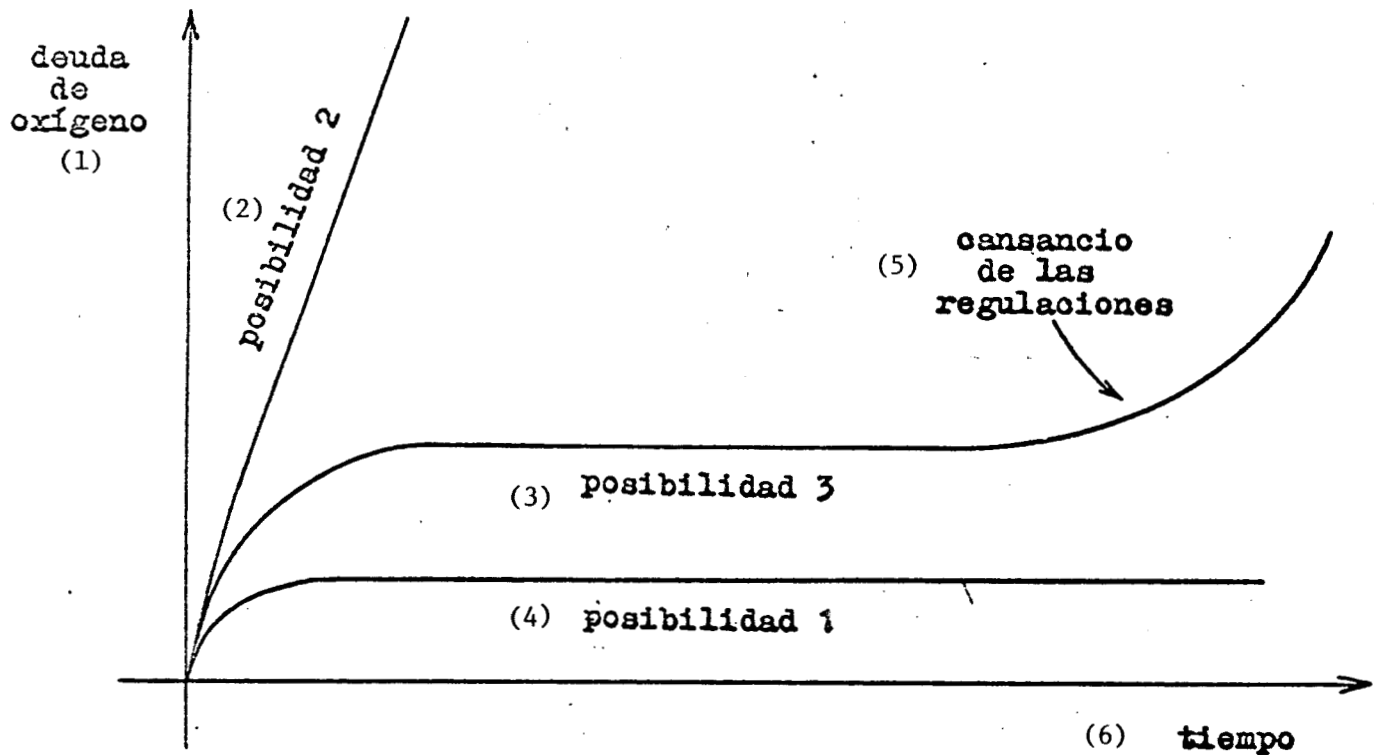controlled in accordance with this possibility.

Figure 1

Possibilities for the Behavior of the Oxygen Debt of the Heart,
When the Cardiac Oxygen-Energy Equilibrium is Disturbed.
The disturbance duration is indefinite.

(1)- Oxygen Debt; (2)- Possibility 2; (3)- Possibility 3;
(4)- Possibility 1; (5)- Fatigue of Controls; (6) - Time.

If, on the other hand, the virtual unevenness reaches very high values,

the regulatory mechanisms are not able to re-establish the equilibrium

between both elements of (I). As a consequence, the oxygen debt of the

heart will increase in a progressive manner, just as occurs in the case of

progressive hemorrhage shock (Ref. 4).

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But there is still another possibility. This possibility consists of the fact that the virtual unevenness is greater than the mentioned threshold in the first possibility, but not sufficiently large to cause the second possibility. In this case, once the already-mentioned initial oxygen debt is contracted, the controls can compensate for the virtual unevenness, but not for the entire period of time - which, in principle, is indefinitely long - which the disturbance can last. This is due to the fact that, when the body controls should be in operation at a certain intensity, they cannot all last for an indefinitely long period of time. This is caused by the fact that the nervous system and other compensatory mechanisms are subject to fatigue. In this case, depending upon how much time elapses, /7 some body controls will operate with less efficiency, or will stop operating completely. As a consequence, if the disturbance continues, the time will arrive when the reduced controls can no longer avoid the appearance of an effective unevenness between both elements of (I). When this occurs, the oxygen debt of the heart will begin to increase in a progressive form, which will lead to the death of the body.

Chapter IV

Cardiac Oxygen-Energy Equilibrium in the Absence of Gravity

The existence of suction in part of the ventricle during diastole was demonstrated experimentally by G. A. Brecher (Ref. 16). After setting up an appropriate valve arrangement, this author introduced a canula into the ventricle. Using a deligation, he obstructed the auricle-ventricle opening and observed that - when the canula reached a deposit of water located at a level which was lower than that of the heart - the diastolic activity

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emptied the deposit. There is no complete accord regarding the causes

determining the suction. This fact could be due, according to Cignoline,

to coronary flow. According to Villa, it could be due to cardiac decon-

traction; according to Torrent - Gausp (Ref. 17), it could be due to con-

traction of endocardiac fibers. The authors overlooked the hypothesis

that at least one part of the diastolic ventricular suction is due to a

small drop of blood contained in the ventricles during the diastolic period.

In the case of man, at the beginning of the diastole there are approximately

250 grams of blood in the ventricles (Ref. 10), as opposed to some 300 grams

which the empty myocardium of a normal adult weighs.

The weight of the intracardiac blood would contribute to expansion of

the ventricles during the diastolic period. Therefore, a certain portion

of the **decrease** observed in the ventricular pressure in the diastole could

be due to this expansion. In the absence of gravity, the intracardiac blood

has no weight, and - when one of the factors disappears which normally con-

tributes to expansion of the ventricles - the diastolic pressure increases.

The increase in the diastolic intraventricular pressure causes, in its turn,

a decrease in the ventricular suction effect during the diastole. In the

absence of gravity, this decrease in the diastolic ventricular suction pro-

duces the following additional factors of decompensation of the cardiac

oxygen-energy equilibrium, in relation to the decumbent position.

(We shall refer all of these changes to the decumbent position, and not

to the erect position, since we are interested in comparing the virtual un-

evenness of the cardiac oxygen-energy equilibrium in the decumbent position

with the virtual unevenness in the absence of gravity.)

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(1) Due to an increase in the diastolic intraventricular pressure, the venous return of the blood which has irrigated the heart is rendered more difficult. This difficulty affects both the blood which returns by the /8 Thebesian conduits, as well as the blood which returns by the coronary cavity.

(2) Owing to the decrease of the cardiac suction, the right intraauricular pressure should increase somewhat. Since very small changes in the right intraauricular pressure significantly change the venous return which passes through the venae cavae (Ref. 18), a decrease in the venous return will be produced in the absence of gravity. The decrease in the venous return produces, due to the vagal reflex already mentioned in Chapter II, an obstruction of the coronary circulation in relation to the decumbent position. Under conditions of weightlessness, it has been effectively verified that the venous return is obstructed in mammals (Ref. 19).

In order to compare the virtual unevenness of the equilibrium (I), which occurs in the absence of gravity, with that which is produced in the decumbent position, it is necessary to study the manner in which the incidence varies – over said equilibrium – of the factors which have already been studied in the decumbent position.

The pressure in the carotid sinus is modified in a manner which is very similar to that which occurs in the decumbent position. As a consequence, the reflex which leaves the carotid sinus will not modify its influence upon the virtual unevenness of the cardiac oxygen-energy equilibrium, when one passes from the decumbent position to the absence of gravity.

The variation in the volemia will be at least equal to that which is produced in the decumbent position. Although a variation in the hemoglobin concentration in relation to the decumbent position cannot be inferred from this

fact, in combination with the statements given in (2), it will give way to the following decompensation factor.

(3) In the absence of gravity, the given volemia increase is related to a volume per minute which is less than that which exists in the decumbent position. Therefore, based on the statements given in Chapter II, in this case stress-relaxation which is much more intense than that occurring in the decumbent position should be produced, with the following, more exaggerated decrease in the diastolic pressure (Ref. 20).

Naturally, the complex regulations which compensate for the virtual unevenness of the equilibrium (I) can operate in many ways, including the modification of variables whose value has been changed by the action of a given decompensation factor. Therefore, it is possible that the effective decrease in the diastolic pressure does not totally reflect the degree of stress-relaxation which is reached in many areas of the body, for the reasons given in (3).

In relation to the decumbent position, as a compensation factor we have the lesser cardiac action which results principally from a decrease in the volume per minute (the decrease in the diastolic pressure can be neutralized, in relation to the cardiac action, by an increase in the systolic pressure [Ref. 20]).

The vagal reflex mentioned in Chapter II tends to adapt the coronary circulation to variations in the cardiac load caused by changes in the volume per minute. The authors assume that the consistent compensation factor and /9 the decrease of the volume per minute is approximately neutralized by the decompensation factors given in (2). If this were the case, the virtual unevenness of the cardiac oxygen-energy equilibrium would be greater, in the

absence of gravity, than the virtual unevenness of said equilibrium in the decumbent position, owing to the incidence of the factors (1) and (3) already mentioned.

An increase in the virtual unevenness of the cardiac oxygen-energy equilibrium opens up the possibility that the body controls which compensate for the given virtual unevenness cannot operate indefinitely. In other words, if the virtual unevenness of the cardiac oxygen-energy equilibrium is greater than a certain critical threshold value, we come face to face with the third possibility mentioned in Chapter III - in other words, we come face to face with the combination of the second and third possibilities presented in that chapter. Naturally, it cannot be stated absolutely that the virtual unevenness of the cardiac oxygen-energy equilibrium exceeds the critical threshold value which was mentioned in the absence of gravity. Nevertheless, based on the statements which have been presented in this chapter, we cannot discard this alternative. In the next chapter we shall analyze what would occur in this case.

Chapter V

Possible Behavior of the Heart in the Absence of Gravity

The insufficient contribution of a substance which is as essential as oxygen sets into operation several vascular mechanisms with the purpose of compensating for this. According to (Ref. 10), "These mechanisms are, naturally, the same ones which intervene in order to increase the blood flow to the tissues when their activity increases. If it is necessary to utilize appropriate mechanisms in order to permit a supplementary consumption during exertion, in order to compensate for the pathological decrease of the sanguine

contribution under basal conditions, the maximum limit of the exertion will suffer a proportional loss." In our case, we are not dealing with a pathlogical phenomenon, but an external disturbance - the absence of gravity - causing a reduction in the blood carried to the heart, by means of the same physiological mechanisms of the body. On the basis of the statements given above, it can be seen that the effort produced by the body controls can be translated not only into the possibility of an increase in the intensity of a function performed by this body, but it can also be translated into the possibility of the basal operation of the body, or of an organ, under unfavorable conditions. We shall call this latter type of effort basal effort.

The latter phenomenon occurs when the heart operates in the absence of gravity. As a consequence, the maximum effort which - with the same training - a heart can perform will decrease in the absence of gravity. For this reason, individuals which are not trained will encounter difficulty in performing normal efforts in the absence of gravity. If a decrease in   /10 the cardiac size is observed in the absence of gravity, it could be caused both by a decreased volume per minute, or by a miocardia (Ref. 10) caused by the basal effort which the heart is performing.

All of the preceding considerations refer to the occurrences taking place during the interval in which the body controls can compensate for and annul the deficit in the oxygen transported to the heart.

Once a sufficient amount of time has elapsed in the absence of gravity, the given body controls begin to tire - in accordance with the statements made at the end of the preceding chapter. Due to this fact, they can no longer block the existence of an effective lack of equilibrium between the oxygen contribution and the cardiac operation. In other words, they can not

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stop the virtual unevenness of the cardiac oxygen-energy equilibrium from

becoming a real unevenness. Since the heart performs, within certain

limits, the work required by general circulation - even though the oxygen

contribution is not sufficient to do this - it will be obliged to contract

an oxygen debt which is greater each time. Once this exceeds certain

limits, it begins to destroy the contractile capacity of the heart (Ref. 21)

which would lead, by means of a vicious circle, to the death of the body.

If we assume, along with A. C. Guyton and J. W. Crowell (Ref. 22), that

cardiac deterioration is the essential characteristic of circulatory

shock, it can be stated that this death will be due to shock - although

there were no previous hypervolemia.

Let us now analyze the possibility of symptoms which may reveal the

appearance of this cardiac deterioration. Given the origin of the oxygen

debt, it is to be hoped that the cardiac anoxia will be uniform throughout

the entire heart. But according to (Ref. 10), "the heart which is uniformly

anoxic does not perform fibrillation, even though showing intense cyanosis,

and the electrocardiograph and vectocardiograph recordings of it are identi-

cal to those of a heart which is oxydized normally".

Since the shock is produced in this case without changes in the volemia,

we are confronted with a case which is similar to that which occurs when -

in the irreversible phase of progressive hemorrhagic shock - a blood trans-

fusion is performed to recover the lost volemia. These experiences show

(Ref. 23) that the volume per minute, as well as the pressure (Ref. 24), begin

to decrease in a significant manner in the irreversible phase of shock.

As a consequence, during the reversible phase of this type of shock,

neither the electrocardiograph recording nor the arterial pressure recording

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supply information regarding cardiac deterioration. In order to obtain

this information, the fact must be taken into account that – as the shock

progresses – the curves which show the cardiac effectiveness are displaced

to the right (Figure 2). As a consequence, in conformity with the volume

per minute (horizontal line in Figure 2) the auricular pressure will increase.

Due to this fact, the venous pressure will increase concommitantly. If we   /11

extrapolate the quantitative data obtained for dogs to the case of man

(Figure 2), it can be calculated approximately that, during the reversible

phase of shock, the right auricular pressure along with the venous pressure

will increase by about 4 mm Hg. In accordance with these data, a

further increase in the venous pressure implies the possibility of an irre-

versible cardiac deterioration.

The time intervals which designate distinct stages in hemorrhagic shock

cannot be compared with the time intervals which designate identical stages

in this type of shock occurring in the absence of gravity. The reason for

this lies in the fact that the phenomena which occur in progressive hemorrhagic

shock develop in accordance with the second possibility given in Chapter III –

i.e., much more rapidly. Nevertheless, if a slow increase in the venous

pressure is noted during the absence of gravity, it will be an indication

that in the absence of gravity the heart behaves in accordance with the state-

ments presented here.

In order to terminate this chapter, we shall study the effects of physical

exercise in the absence of gravity.   /12

Exercise performed in the absence of gravity, like any bodily condition

which requires major work by the heart, implies the necessity of great coronary

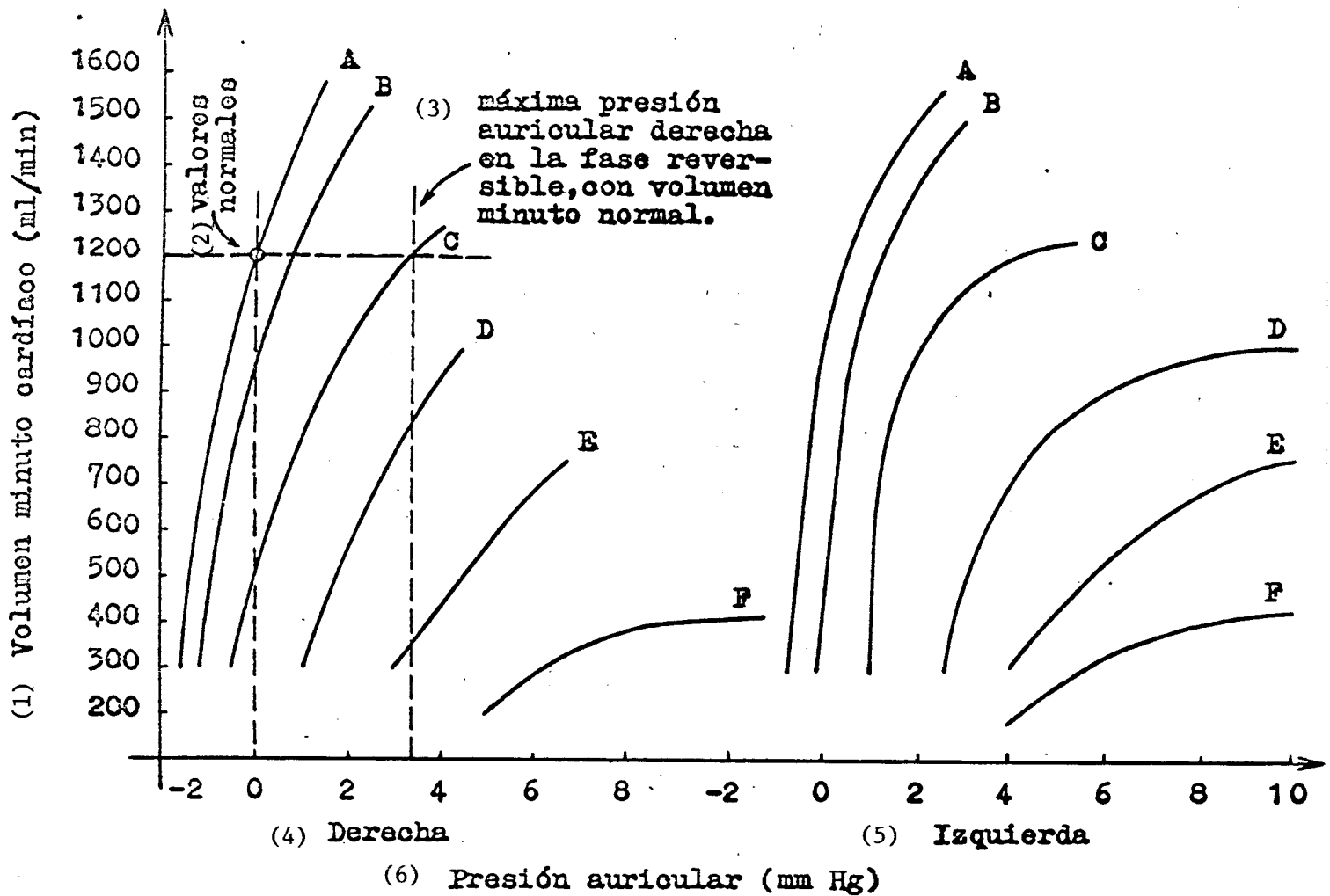circulation. This will be achieved by the intervention of the body

Figure 2

Progressive Deterioration of the Cardiac Expulsion Curves in
Progressive Hemorrhagic Shock. A: normal curves; B and C: curves
during the reversible phase; D, E, and F: curves in the irreversible
phase. The time scale is not comparable with that which occurs in the
absence of gravity (see the text). Modification of J. W. Crowell
and A. C. Guyton, 1962.

(1) – volume per minute (ml/min); (2) – normal values; (3) – maximum
right auricle pressure in the reversible phase, with normal volume per
minute; (4) – right; (5) – left; (6) – auricular pressure (mm Hg).

controls. But if these controls are insufficient, in the

absence of gravity, to maintain the basal cardiac state during an indefinite

time – no matter what additional effort is required of them – the development of fatigue will only be accelerated. The acceleration of fatigue in the controls will shorten the interval during which the controls can compensate for the effects produced by the absence of gravity upon the coronary circulation, thereby advancing the moment at which a constant increment is produced in the cardiac oxygen debt. As a consequence, exercise performed during the absence of gravity is prejudicial for the heart.

This exercise must be differentiated from exercise performed periodically before an orbital flight which, when the individual is trained, increases his regulatory capacity and therefore his capacity to resist the absence of gravity.

Chapter VI

Orientation of Methods to Correct and Prevent Cardiac Deterioration

in the Absence of Gravity

The methods which are employed in the absence of gravity can be divided into two groups: (1) those whose purpose is to impede irreversible cardiac deterioration (corrective methods); and (2) those whose purpose is to impede total cardiac deterioration (preventive methods). In accordance with the statements already presented, a simple corrective method would be as follows. The venous pressure of the astronaut is measured continuously. If it begins to advance to a basal state or manifests abnormal behavior during exercise, the astronaut should be digitalized by means of strophantin. The digitalization increases the oxygen debt which the heart can support without suffering irreversible harm. This would provide time enough to effect the decrease and time enough for the astronaut to recover before his cardiac deterioration became irreversible.

Due to the fact that we have no knowledge of the curves for cardiac effectiveness in man during progressive shock, as well as the value of the volume per minute in the absence of gravity, we can make no categorical statements regarding the quantitative value of the increment in auricular pressure and venous pressure during the reversible phase of cardiac deterioration. If the venous pressure is measured instead of the auricular pressure, the fact must be taken into account that the venous pressure can be affected by other factors. In accordance with the data obtained for animals, it can be assumed that if an increase of 15 to 20 mm of water is observed in the right auricular pressure, the flight should be discontinued. It is most advisable to measure the pressure as close as possible to the right auricule in order to detect as soon as possible any anomaly which cannot be attributed to other causes.

Different techniques can be used as preventive methods, which in turn /13 can be classified in two groups: (1) methods which succeed in abolishing or diminishing the virtual unevenness of the cardiac oxygen-energy equilibrium in the absence of gravity, and (2) methods which succeed in replacing or helping the body controls, thus impeding the fatigue of these controls.

A. <u>Preventive Methods of the First Group.</u>

Due to the fact that a large portion of the virtual unevenness of the cardiac oxygen-energy equilibrium is caused by vagal reflexes, an attempt could be made to block the action of the vagus in the absence of gravity by means of atropine. The administration of atropine could prevent cardiac deterioration, but it is apparent that the astronaut is thus subjected to an extremely undesirable handicap.

The theory formulated by the authors relative to ageing (see the attached

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report) predicted the existence of substances which could replace atropine, without causing blockage of the vagus but increasing the physiological permeability of the cardiac capillaries and of the cardiac cells. In this way, there would be a change in the point of equilibrium toward which the body controls, as well as the vagal control, tend. Thus, the virtual unevenness of the cardiac oxygen-energy equilibrium could be reduced without causing other undesirable secondary effects. Due to the fact that the practical application of this type of substance requires biochemical and biological investigations which cannot be carried out rapidly, we cannot go into this question in greater detail until it has been verified that the cardiac behavior in the absence of gravity is effectively the same as that which has been presented in this article.

B. **Preventive Methods of the Second Group**.

It is apparent that the body controls are assisted when the coronary circulation is facilitated mechanically. We are naturally referring to the effective coronary discharge which circulates through the cardiac capillaries, and not to the coronary discharge circulated through the arterioluminar canals.

A bloodless way to achieve this objective could consist of the iron heart developed by Dr. M. M. Nachlas and the Westinghouse Company. This heart is capable of actuating the blood when the heart is obstructed by means of external compression and decompression of the chest. Although this apparatus has never been used in hearts operating physiologically, we have consulted Dr. M. M. Nachlas, and he is of the opinion that there is a possibility that the apparatus can, by means of adequate synchronization, increase the force of the blood in a heart which is operating. It is

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apparent that if the force of the cardiac systole increases, other conditions being equal, the coronary discharge will also increase. The principal drawback of this method is its quantitative limitations, since the rigidity of the cardiac wall will probably impede a large portion of the energy which the apparatus can supply. If for this reason the energy transmitted is insufficient, it would be necessary to employ methods entailing blood.

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